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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.

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Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

SEP - 7 2004
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U.S. PROSECUTION

Office Action Summary

Application No.	Applicant(s)
09/445,517	Duft et al.
Examiner	**Group Art Unit**
S. Devi, Ph.D.	1645

☒ Responsive to communication filed on *02/05/01.*

☐ This action is **FINAL.**

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire *three* month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

Disposition of Claims

☒ Claim(s) *1-15* is/are pending in the application.

Of the above, claim(s) _____ is/are withdrawn from consideration.

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) *1-15* is/are rejected.

☐ Claim(s) _____ is/are objected to.

☐ Claims _____ are subject to restriction or election requirement.

Application Papers

☐ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The proposed drawing correction, filed on _____ is ☐approved ☐disapproved.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).

☐ All ☐ Some* ☐ None of the CERTIFIED copies of the priority documents have been

☐ received.

☐ received in Application No. (Series Code/Serial Number) _____.

☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

*Certified copies not received: _____.

☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

☒ Notice of References Cited, PTO-892

☐ Information Disclosure Statement(s), PTO-1449, Paper No(s). _____

☐ Interview Summary, PTO-413

☐ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Notice of Informal Patent Application, PTO-152

--- SEE OFFICE ACTION ON THE FOLLOWING PAGES ---

DETAILED ACTION

Continued Prosecution Application

1) The request filed on 02/05/2001 (paper no. 5) for a Continued Prosecution Application (C.P.A) under 37 C.F.R 1.53(d) based on parent Application, SN 09/445,517, is acceptable and a C.P.A has been established. An action on the C.P.A follows.

Status of Claims

2) Claims 1-15 are pending in the instant application and are under examination. A First Action on the Merits is issued for these claims.

Prior Citation of Title 35 Sections

3) The text of those sections of Title 35 U.S. Code not included in this action can be found in a prior Office Action.

Prior Citation of References

4) The references cited or used as prior art in support of one or more rejections in the instant Office Action and not included on an attached form PTO-892 or form PTO-1449 have been previously cited and made of record.

Priority

5) The instant application is a 371 of PCT/US98/11753 filed 06/05/1998, which is a Continuation-in-part of application, SN 09/870,762, filed 06/06/1997, which is *now co-pending.*

Objection(s) Maintained

6) The objection to the specification made in paragraph 4 of the Office Action mailed 08/04/00 (paper no. 3) with regard to the missing abstract, is maintained for reasons set forth therein.

7) The objection to the specification made in paragraph 5 of the Office Action mailed 08/04/00 (paper no. 3) is maintained for reasons set forth therein.

Rejection(s) Withdrawn

8) The rejection of claims 1 and 2 made in paragraph 8 of the Office Action mailed 08/04/00 (paper no. 3) under 35 U.S.C § 102(b) as being anticipated by Cooper *et al.* (US 5,280,014) ('014), or Cooper *et al.* (US 5,364,841) ('841) is withdrawn.

9) The rejection of claims 1-3 made in paragraph 9 of the Office Action mailed 08/04/00 (paper no. 3) under 35 U.S.C § 102 (e) as being anticipated by Rink *et al.* (US 5,739,106) [Rink *et al.* ('106)] is withdrawn.

10) The rejection of claims 1-10 made in paragraph 11 of the Office Action mailed 08/04/00 (paper no. 3) under 35 U.S.C § 103(a) as being unpatentable over Rink *et al.* (US 5,739,106) [Rink *et al.* ('106)] in view of Gaeta *et al.* (US 5,686,411) is withdrawn.

11) The rejection of claims 1-10 made in paragraph 12 of the Office Action mailed 08/04/00 (paper no. 3) under 35 U.S.C § 103(a) as being unpatentable over Kolterman *et al.* (*Diabetologia* 39: 492-499, April, 1996) (Kolterman *et al.*, 1996), or Kolterman *et al.* (WO 96/40220) ('220), or Moyses *et al.* (*Diabetic Med.* 13 (suppl. 5): S34-S38, September, 1996), or Thompson *et al.* (*Diabetes* 46: 632-636, April 1997) in view of Cooper *et al.* (*Biochim. Biophys. Acta* 1014(3): 247-258, 1989, abstract) (Cooper *et al.*, 1989) and Rink *et al.* (US 5,739,106) [Rink *et al.* ('106)] is withdrawn.

Rejection(s) Maintained

12) The rejection of claims 1-10 made in paragraph 6 of the Office Action mailed 08/04/00 (paper no. 3) under 35 U.S.C § 112, first paragraph, as being non-enabled with regard to the scope, is maintained for reasons set forth therein.

12) The rejection of claims 11-15 made in paragraph 13 of the Office Action mailed 08/04/00 (paper no. 3) under 35 U.S.C § 103(a) as being unpatentable over Kolterman *et al.* (*Diabetes Care* 18: 1179-1182, August 1995) (Kolterman *et al.*, 1995) in view of Rosenbloom *et al.* (*Am. J. Dis. Child.* 131: 881-885, 1977), Rink *et al.* (WO 92/20367) (Rink *et al.*, '367) and Morley *et al.* (*Am. J. Physiol.* 267: R178-R184, 1994) (Morley *et al.*, 1994) is maintained and is reiterated herebelow.

Kolterman *et al.* (1995) teach the administration of an amylin species, ^{25,28,29}pro-h-amylin, and amylin agonist therapy in patients with type 1 diabetes mellitus taking insulin (see page 1181, column 3 and page 1179, last paragraph). Kolterman *et al.* are silent about the status of insulin-induced weight gain in the type 1 diabetic patients that they treated and the effect of amylin or amylin agonist on weight gain.

However, Rosenbloom *et al.* teach that insulin overdosage in type 1 diabetic patients leads to excessive appetite and weight gain (see abstract).

Rink *et al.* ('367) disclose that amylin can act as an appetite suppressant (see page 11).

Morley *et al.* (1994) teach a method of reducing food intake in obese and diabetic subjects by administering upto 200 micrograms of amylin per kg (see abstract and page R179). Morley *et al.* (1994) also teach that amylin acts as a peripherally acting satiety agent (see abstract).

Given the art-recognized association between insulin overdosage in IDDM and excessive appetite and weight gain as taught by Rosenbloom *et al.*, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to use Kolterman's (1995) method of treatment with an amylin species, i.e., $^{25, 28, 29}$pro-h-amylin, for type 1 diabetic patients suffering from insulin-induced weight gain, to produce the instant invention, because amylin is known in the art to act as an appetite suppressant as taught by Rink *et al.* ('367). A skilled artisan would have had a reasonable expectation of success in using Kolterman's (1995) method in reducing insulin-induced weight gain in type 1 diabetic patients, since amylin is known to reduce food intake in obese and diabetic subjects as taught by Morley *et al.* (1994).

Claims 11-15, as a whole, are *prima facie* obvious over the prior art of record.

Double Patenting Rejection(s)

13) The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970) and *In re Thorington,* 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 C.F.R 1.321(c) may be used to overcome an actual or provisional rejection based on a non-statutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 C.F.R 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal

disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 C.F.R 3.73(b).

Instant claims are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over the claims of the co-pending application, SN 09/870,762. Although the conflicting claims are not identical, they are not patentably distinct from each other, because of the overlapping scope of the claims.

14) Claims 1-5 are rejected under 35 U.S.C § 103(a) as being unpatentable over Arnelo *et al.* (Arnelo *et al. Am. J. Physiol.* 271: 6 pt 2: R1654-R1659, December 1996) (Arnelo *et al.* I), or Arnelo *et al.* (*Scand. J. Gastroenterol.* 31: 83-89, January 1996) (Arnelo *et al.* II).

It is noted that the claimed method uses a composition comprising either amylin or an amylin agonist. It is also noted that claims 1-5 do not recite any doses of amylin or amylin agonist. Claims 1-3 do not recite any routes of administration. In this rejection, the method of treatment of obesity is viewed as the same as reduction in body weight in light of the experimental results presented in Example 1 of the instant specification.

Arnelo *et al.* (I) teach a method of reducing food intake or decreasing body weight gain, i.e., treating obesity, in rats by subcutaneous administration of a composition consisting of IAPP (i.e., amylin) in a vehicle (i.e., pharmaceutically acceptable carrier) containing saline (see abstract; page R1655 and page R1654, left column, last paragraph). The composition contains 7 or 25 or 50 pmol/kg/min (i.e., an effective amount) of IAPP, which significantly decreased body weight gain (see page R1656, right column; Figure 4, and page R1657, right column).

Arnelo *et al.* (II) teach a method of reducing body weight, i.e., treating obesity, by subcutaneous administration of 50 pmol/kg/min (i.e., an effective amount) of a composition consisting of IAPP (i.e., amylin) and a DMSO-containing saline to rats (see page 85, left column; page 84, both columns and abstract). The prior art method significantly decreased the body weight and food intake in amylin-treated rats (see page 86 and Figure 4). Arnelo *et al.* (II) further teach IAPP or amylin to be a satiety factor (see page 87, left column).

Arnelo *et al.* (I) or Arnelo *et al.* (II) differ from the instant invention in not using their method for decreasing weight gain or reducing food intake or treating obesity in a human subject.

However, a method of suppressing food intake in a subject using a compound can reasonably be viewed by one skilled in the art as a method of treating obesity, since a skilled

artisan would understand that suppression of food intake would lead to loss of body weight. Further, given that the rat model is widely accepted in the art as predictive of weight reduction in humans, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to use or extend Arnelo's (I or II) method of decreasing body weight gain to another mammalian subject, such as a human subject, to produce the method of the instant invention, with a reasonable expectation of success. Since human clinical trials are often conducted following successful animal experimentation, a skilled artisan would have been motivated to produce the instant invention by extending Arnelo's method (I or II) to humans for the expected benefit of reducing the incidence of obesity in humans, as treating obesity in humans is highly desired in the art.

A specific secondary reference teaching that animal models are generally accepted in the art as predictive of reduction in weight gain or obesity, or suppression of food intake in humans, is **not** applied in this rejection, since such a teaching is widely known to those skilled in the art. See the section 'Prior Art' below for animal models widely accepted in the art.

Claims 1-5 are *prima facie* obvious over the prior art of record, absent evidence to the contrary.

15) Claims 6-8 are rejected under 35 U.S.C § 103(a) as being unpatentable over Arnelo *et al.* (Arnelo *et al. Am. J. Physiol.* 271: 6 pt 2: R1654-R1659, December 1996) (Arnelo *et al.* I), or Arnelo *et al.* (*Scand. J. Gastroenterol.* 31: 83-89, January 1996) (Arnelo *et al.* II) as applied to claim 5 above, and further in view of Bennett *et al.* (US 5,955,443).

The teachings of Arnelo *et al.* (I) or Arnelo *et al.* (II) modified as explained above do not disclose the use of 30-300 microgram or 60 microgram/dose of amylin 1 to 4 times a day.

However, effective doses of a pharmaceutical compound and optimal frequency of its administration to a human subject can be readily determined by routine experimentation by those skilled in the art based on the age, sex, weight, clinical condition and extent of a clinical condition in a human subject, or can be determined by a medical practitioner on a case by case basis. Nothing more than a routine skill is needed for such a determination. For example, Bennett *et al.* teach that (see column 27, lines 32-45):

> Human doses can be extrapolated from animal studies (Katocs et al., Chapter 27 In: Remington's

> Pharmaceutical Sciences, 18th Ed., Gennaro, ed., Mack Publishing Co., Easton, Pa., 1990). Generally, the dosage required to provide an effective amount of a pharmaceutical composition, which can be adjusted by one skilled in the art, will vary depending on the age, health, physical condition, weight, type and extent of the disease or disorder of the recipient, frequency of treatment, the nature of concurrent therapy (if any) and the nature and scope of the desired effect(s) (Nies et al., Chapter 3 In: Goodman & Gilman's The Pharmacological Basis of Therapeutics, 9th Ed., Harman et al., eds., McGraw-Hill, New York, N.Y., 1996).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to arrive at the dose and the frequencies of administration of amylin in humans, as recited in instant claims, by routine experimentation or optimization, for use in Arnelo's (I) or Arnelo's (II) method as modified and produce the method of the instant invention.

Claims 6-8 are *prima facie* obvious over the prior art of record.

16) Claims 1-10 are rejected under 35 U.S.C § 103 (a) as being unpatentable over Kolterman *et al.* (WO 96/40220, already of record) (Kolterman *et al.* II) in view of Meglasson (US 5,134,164).

It is noted that Example 1 of the instant specification enables the claimed method for reducing weight in patients having type II diabetes mellitus.

Kolterman *et al.* (II) teach a method of treating type II diabetes mellitus (i.e., NIDDM) comprising administering a therapeutically effective amount of an amylin agonist, such as, ^{25,28,29}pro-h-amylin and h-amylin. Administration is preferably by subcutaneous injection (abstract and claims). Amylin agonists, such as, ^{25,28,29}pro-h-amylin, may be administered in single or multiple doses, for example, two (BID), three (TID), and/or four (QID) times per day. BID doses range from about 30 μg to 150 μg BID, more preferably from about 50 μg to 60 μg BID. TID doses range from about 30 μg to 150 μg, more preferably about 60 μg TID. QID doses range from about 30 μg to 60 μg QID, more preferably about 30 μg QID. These doses have been demonstrated to be effective in various human clinical trials and are administered subcutaneously (see page 21). Kolterman *et al.* teach that type II diabetes mellitus is characterized by hyperglycemia (see page 7, lines 12 and 13) and that AC137 (i.e., ^{25,28,29}pro-h-amylin) induces reduction in hyperglycemia in type II diabetic patients (see claims 2, 7 and 11, and pages 12-14).

Kolterman *et al.* (II) do not expressly state that their method is also useful in the treatment of obesity.

However, that Kolterman's (II) method also serves as a method of treatment of obesity is implicit from the teachings of Kolterman *et al.* (II) in light of what is well known in the art. For instance, Meglasson discloses that hyperglycemia occurs in obesity and non-insulin dependent diabetes mellitus (NIDDM) (see column 1, third paragraph). It is taught that excess adiposity can be seen in NIDDM associated with obesity and obesity without NIDDM (see column 2, first sentence). Most importantly, Meglasson explicitly teaches that a compound that is useful in the treatment of hyperglycemia "could also be used to treat or prevent NIDDM" and "**obesity**" (see column 2, lines 21-25).

Given that amylin agonist, ^{25,28,29}pro-h-amylin, has already been identified in the art as a compound that is useful in the treatment of hyperglycemia and NIDDM, or as a compound that reduces hyperglycemia in patients with type II diabetes, i.e., NIDDM, as taught by Koltermann *et al.* (II), it would have been obvious to one of ordinary skill in the art at the time the invention was made to use Kolterman's (II) method of reducing hyperglycemia for treating obesity to produce the instant invention, with a reasonable expectation of success, because Meglasson explicitly teaches that any compound that is useful in the treatment of hyperglycemia could also be used to treat or prevent obesity. A skilled artisan would understand that Kolterman's anti-hyperglycemic compound, ^{25,28,29}pro-h-amylin (i.e., an amylin agonist analogue), would also serve as an anti-obesity agent. Since there is an art-recognized need for reducing the incidence of human obesity in general and/or in diabetic population, one skilled in the art would have been motivated to use Kolterman's method of reducing hyperglycemia in humans to treat obesity for the expected benefit of reducing the increasing incidence of obesity, because Meglasson explicitly teaches that any compound that is useful in the treatment of hyperglycemia could also be used to treat or prevent obesity.

Claims 1-10 are *prima facie* obvious over the prior art of record.

17) Claims 1 and 5 are rejected under 35 U.S.C § 103 (a) as being unpatentable over Morley *et al.* (*Am. J. Physiol.* 267: R178-R184, 1994) (Morley *et al.*, 1994).

Morley *et al.* teach a method of effectively suppressing or reducing food intake (i.e., treating obesity) in genetically obese (*ob/ob*) and lean (*ob/c*) mice as well as in diabetic (*db/db*) and lean (*db/c*) mice by administering upto 200 micrograms of amylin per kg (see abstract and

page R179). Morley *et al.* (1994) also teach that amylin acts as a peripherally acting satiety agent (see abstract). Morley *et al.* (1994) further disclose that the obese mouse (*ob/ob*) is a classical genetic model of obesity (see page R178, right column, first full paragraph).

Morley *et al.* (1994) differ from the instant invention in not using their method for suppressing or reducing food intake or treating "obesity" in a human subject.

However, a method of suppressing food intake in a subject using a compound can reasonably be viewed by one skilled in the art as a method of treating obesity, since a skilled artisan would understand that suppression of food intake would lead to loss of body weight. Further, given that the murine model is widely accepted in the art as a classical model of obesity as taught by Morley *et al.* (1994) and as predictive of weight reduction in humans, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to use or extend Morley's method of suppressing or reducing food intake to another mammalian subject, such as a human subject, to produce the method of the instant invention, with a reasonable expectation of success. Since human studies are often conducted following successful animal experimentation, a skilled artisan would have been motivated to produce the instant invention by extending Morley's to humans for the expected benefit of reducing the incidence of obesity in humans, as treating obesity in humans is highly desired in the art.

Claims 1 and 5 are *prima facie* obvious over the prior art of record.

Relevant Prior Art

18) The prior art made of record and not currently relied upon in any of the rejections is considered pertinent to Applicants' disclosure:

- Kleyn *et al.* (US 6,043,346) disclose that obesity poses a major worldwide health problem (see column 4, lines 22 and 23). Kleyn *et al.* teach that animal-based body weight disorder systems, i.e., tub mice models, may be used to identify compounds capable of ameliorating body weight disorder-like symptoms. Such animal models may be used as test substrates for the identification of drugs, pharmaceuticals, therapies and interventions which may be effective in treating such disorders. For example, animal models may be exposed to a compound, suspected of exhibiting an ability to ameliorate body weight disorder symptoms, at a sufficient concentration and for a time sufficient to elicit such an amelioration of body weight

disorder symptoms in the exposed animals. The response of the animals to the exposure may be monitored by assessing the reversal of disorders associated with body weight disorders such as obesity. With regard to intervention, any treatments which reverse any aspect of body weight disorder-like symptoms should be considered as candidates for human body weight disorder therapeutic intervention. Dosages of test agents may be determined by deriving dose-response curves (see column 32, fourth full paragraph).

- Venkatesan (US 6,020,361) teaches compounds which show much higher antiobesity and antihyperglycemic activity in animal models and conclude that such compounds are useful in treating diabetes, hyperglycemia, and obesity in humans and animals, when formulated into pharmaceutical compositions (see column 2, lines 10-24).

- Korsgaard *et al.* (US 6,008,242) teach a rabbit model of obesity and state that it is a generally recognized model of obesity. The data obtained with an anti-obesity agent in a rabbit model are taught to be useful in using the agent as a therapeutic agent against obesity in mammals, including primates such as humans (see column 2).

- Tartaglia *et al.* (US 5,972,621) teach that animal-based body weight disorder systems, which may include, for example, *ob*, *db* and *ob/db* mice, may be used to identify compounds capable of ameliorating body weight disorder-like symptoms. Such animal models may be used as test substrates for the identification of drugs, pharmaceuticals, therapies and interventions which may be effective in treating such disorders. For example, animal models may be exposed to a compound, suspected of exhibiting an ability to ameliorate body weight disorder symptoms, at a sufficient concentration and for a time sufficient to elicit such an amelioration of body weight disorder symptoms in the exposed animals. The response of the animals to the exposure may be monitored by assessing the reversal of disorders associated with body weight disorders such as obesity. With regard to intervention, any treatments which reverse any aspect of body weight disorder-like symptoms should be considered as candidates for human body weight disorder therapeutic intervention. Dosages of test agents may be determined by deriving dose-response curves (see column 38).

- Lee *et al.* (US 5,932,779) teach a method of identifying compounds for ameliorating body weight disorders by testing in animal model systems for body weight. Lee *et*

al. teach that animal models may be used as test substrates for the identification of drugs, pharmaceuticals, therapies and interventions which may be effective in treating such disorders. Lee *et al.* teach that animal models may be exposed to a compound, suspected of exhibiting an ability to ameliorate body weight disorder symptoms, at a sufficient concentration and for a time sufficient to elicit such an amelioration of body weight: disorder symptoms in the exposed animals. The response of the animals to the exposure may be monitored by assessing the reversal of disorders associated with body weight disorders such as obesity. With regard to intervention, Lee *et al.* teach that any treatments which reverse any aspect of body weight disorder-like symptoms should be considered as candidates for human body weight disorder therapeutic intervention. Dosages of test agents may be determined by deriving dose-response curves (see abstract and column 16).

- Shuldiner *et al.* (US 5,877,283) disclose a method for treatment of obesity and type II diabetes mellitus. Shuldiner *et al.* identify obesity as a primary health concern amongst industrialized countries (see abstract and column 1, last paragraph).

- Taylor *et al.* (US 5,830,434) disclose preferred rat and mouse animal models of hyperglycemia and obesity which could be used for screening for the efficacy of various compounds to treat NIDDM (see column 7, first full paragraph). Taylor *et al.* further disclose how one of ordinary skill in the art can determine the dosage of these compounds (see column 6, lines 10-16):

> The exact dosage may vary on the basis of the patient's age, weight, size and general overall condition and a physician would best be able to determine the exact dosage according to these parameters. Further guidance on determining dosages and modes of administration are available as provided in Remington's Pharmaceutical Sciences (13).

- Svec *et al.* (US 5,527,788) teach obesity as a major health problem of the Western world having medical and economic importance (see column 1, lines 24-28). Svec *et al.* disclose a rat model of obesity which shares many characteristics with human obesity, including hyperglycemia and insulin resistance (see column 7, lines 33-39).

- Wilkison *et al.* (US 6,100,047) disclose that obesity and diabetes animal models can be used to determine the relevance of selective molecules in treating human obesity (see column 4, first full paragraph).

The following prior art references teach that NIDDM is frequently closely associated with obesity :

- Griver *et al.* (*Nutrition Res.* 14: 465-483, 1994) teach that the non-insulin-dependent (NIDDM) form of diabetes is "often" associated with obesity (see abstract).
- Stogdale (*Cornell Vet.* 76: 156-174, 1986) teaches that obesity is "frequently" associated with NIDDM (see abstract).
- Scheen (*Drugs* 54: 355-368, September 1997) teaches that obesity is a condition "frequently" associated with NIDDM (see abstract).
- Johnston *et al.* (*J. Hypertension* 10: 393-397, 1992) teach that obesity and NIDDM are "often associated" (see abstract).
- Thomas *et al.* (*Circulation* 91: 764-770, 1995) teach the association between NIDDM and obesity (see abstract).
- Lutz *et al.* (*Br. Vet. J.*149: 527-536, 1993) teach that obesity is a "frequent concomitant problem" in human Type 2 diabetes (see abstract).
- Meglasson (US 5,134,164) teach that excess adiposity is an etiological factor in NIDDM and when extreme represents a disease state in itself (see abstract).

Remarks

19) Claims 1-15 stand rejected.

20) Papers related to this application may be submitted to Group 1600, AU 1645 by facsimile transmission. Papers should be transmitted via the PTO Fax Center located in Crystal Mall 1. The transmission of such papers by facsimile must conform with the notice published in the Official Gazette, 1096 OG 30, November 15, 1989. The CM1 facsimile center's telephone number is (703) 308-4242, which is able to receive transmissions 24 hours a day and 7 days a week.

21) Any inquiry concerning this communication or earlier communication(s) from the Examiner should be directed to S. Devi, Ph.D., whose telephone number is (703) 308-9347. A message may be left on the Examiner's voice mail service. The Examiner can normally be reached on Monday to Friday from 7.15 a.m to 4.15 p.m. except one day each bi-week which would be disclosed on the Examiner's voice mail system.

If attempts to reach the Examiner by telephone are unsuccessful, the Examiner's supervisor, Lynette Smith, can be reached on (703) 308-3909.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 308-0196.

SD/
S. Devi, Ph.D.
Patent Examiner
April 2001